

**THE
HEALTH EFFECTS
OF
EXPOSURE TO WOOD DUSTS**

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A Study of Nasal and Sinus
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Summary

Human exposure to wood dust may produce a variety of biological effects.

Skin and eye irritation and allergy may be produced by a large variety of woods, primarily tropical species. No dose-response information is available. Wood dust may produce nasal irritation, with discharge or dryness. The respiratory system may be affected, as there is evidence of a minor amount of airways obstruction occurring over a work shift; there is no convincing evidence that permanent airways changes occur, apart from that which occurs in individuals who develop asthmatic reactions to particular woods. In some people allergically-mediated responses are so severe that subsequent exposures to the offending dust must be permanently avoided.

Exposure to wood dusts is convincingly associated with the development of cancer of the nasal cavity and paranasal sinuses. The association has been observed in populations with exposure to either hardwoods or softwoods and the offending agent(s) have not yet been identified. Overall, these cancers are very rare causes of death, and it is estimated that over the past decade, occupational exposure to wood dust has been responsible for less than 1 death per year in the Province of Ontario. There is no convincing evidence that exposure to wood dust increases the risk for any other type of cancer. No dose-response information is available for wood dust and sino-nasal cancer.

Introduction

Many species of wood, both imported and domestic, have commercial uses. Constituents of many woods may produce toxic effects in man. The structural components of wood are cellulose, hemicellulose and lignin. It is believed that chemicals found mainly in the heartwood of the tree are responsible for most toxic effects. Some of these substances act as food reserves, some as hardening agents, and others protect against mechanical injuries or attack by bacteria, fungi, insects, and larger animals. For many of the toxic reactions produced by wood in humans, the responsible agents have not been specifically identified. Some reactions to wood dust, such as cough and sneezing, may be non-specific reactions to dust per se, but others, such as asthma and dermatitis, may be related to sensitivity to the chemical constituents of particular woods. No attempt will be made in this review to discuss every conceivable reaction to the dust of any wood to which Ontario workers might be exposed; rather the health effects of exposure to wood dusts will be discussed in general, with special mention of certain woods when these might be relevant to the health of Ontario workers.

The adverse effects of wood dusts may be divided among several classes:

I Skin and Mucous Membrane Effects

Dermatitis - irritant
 - allergic

Conjunctivitis (Eye inflammation)

Rhinitis (Nasal inflammation)

II Respiratory Effects

Irritant

Asthma

Chronic Airflow Obstruction

III Cancer

Nasal, Sinus

Other Sites

A discussion of these effects is presented in the sections that follow.

I Skin and Mucous Membrane Effects

Dermatitis and Conjunctivitis

The literature dealing with the toxic effects of wood on skin has been extensively reviewed by Woods and Calnan ¹. Toxic woods, nearly always in the form of sawdust, may affect the skin and mucous membranes by direct contact, or constituent chemicals may be absorbed and produce general symptoms. The skin can be damaged by the mechanical action of bristles and splinters, by chemical irritation or sensitization, or by a combination of these.

Irritant chemicals are often found in the outer part of tropical trees and produce their worst effects on timber workers where the trees are felled. The clinical picture is that of an irritant dermatitis with redness and blistering. The conjunctivae of the eyes are also often severely affected.

Sensitizing chemicals (i.e. those producing allergic reactions) are more often found among the extractives of the heartwood and therefore affect sawyers, carpenters and joiners, polishers and finishers exposed to fine dust. The picture is that of an airborne contact dermatitis, beginning on the dorsa of the hands and forearms, eyelids, face and neck and rapidly involving the genitals through handling in micturition. If the clothing gives inadequate protection, fine dust may drift inside and affect the sweaty areas of the axillae, waistband and groin, and sometimes the ankles and dorsa of the feet. Mild cases of dermatitis may show only redness and slight irritation, but often there is a papular or blistering dermatitis which may progress to chronic dermatitis after repeated exposures.

A catalogue of irritating and sensitizing woods is given in Reference 1. Among those which might be commonly encountered in Ontario are Douglas fir, white cedar, beech and oak.

There is no data relating prevalence of skin reactions to airborne dust concentrations. Dust removal by suitable exhaust ventilation and attention to personal hygiene are perhaps the best preventive measures, but once sensitization has occurred avoidance of further contact with the wood responsible might be required.

Rhinitis (Nasal Inflammation)

Wood dust is commonly deposited in the nose, where it may provoke physiological clearance mechanisms such as sneezing and nasal discharge. In addition, certain individuals may develop nasal allergies to various woods (2,3).

Wilhelmsson and Drettner (3) did a cross-sectional epidemiological study of Swedish furniture workers. They found that 20% of exposed workers reported perennial nasal hypersecretion, compared with 12% of slightly exposed or non-exposed workers. In addition, 54% of exposed workers showed abnormally increased nasal mucous transit times as shown by the delay in the passage of dyed mucous through the nose. The mean concentration of wood dust in the air of this factory was 2.0 mg/m³, and in only a few cases was the Swedish TWA of 4 mg/m³ exceeded.

Andersen and colleagues (4) examined 68 Danish furniture workers. The most frequent of the workers' complaints were nasal dryness, eye irritation, nasal obstruction, prolonged colds and frequent headaches; there was no unexposed comparison group. Nasal mucous transit time was increased, and the prevalence of workers with abnormal transit times increased with the dust levels. Nine subjects with abnormal transit times were re-examined at home after a weekend away from work, and in 6 of 9 the transit time had returned to

normal. The authors found that inert plastic dust did not change the transport rate of nasal mucus and concluded that the effect of wood was due to soluble chemical substances in the dust. Boysen and Solberg(5) reasoned that one might be able to detect histological changes in nasal mucosa induced by wood dust exposure. They obtained biopsies from the anterior curvature of the middle turbinate of 113 furniture workers and 54 unexposed control individuals. Metaplastic squamous epithelium and dysplasia were observed more frequently among the furniture workers, as was hyperplastic rhinitis. The metaplastic alterations were considered to be nonspecific, possibly related to age, humidity, dust, and infections, but possibly also to carcinogens. The authors suggested that the areas of dysplasia represent preneoplastic epithelial alteration, but it is not yet known whether these areas might indeed progress to frank malignancy. There was no attempt to correlate histological findings with factory dust levels.

II Effects on Respiratory Function

There are 2 broad categories for the possible effects of wood dust on respiratory function namely, allergic and non-allergic. The wood species most likely to produce allergic (asthmatic) lung responses in workers in Ontario is western red cedar. Dusts of other commonly used woods might, in principle, produce "industrial bronchitis" and airways obstruction, but there is as yet no good evidence that exposure to wood dust leads to chronic non-allergic respiratory changes.

Only a small proportion of the dust generated in wood working operations is in the respirable size range capable of penetrating to the small airways, and total tracheobronchial deposition, for nose breathing, seldom exceeds 10% of the inhaled aerosol mass as very few particles greater than 5 microns aerodynamic diameter will penetrate the nose (6). Hounam and Williams (7) carried out

a dust survey in 5 furniture factories in the United Kingdom and found that sawing operations produced the coarsest dust and sanding the finest. Most of the mass of the airborne dust had an aerodynamic equivalent diameter in excess of 5 microns. Similarly, Holness and colleagues (8) found that much of the dust measured in 4 Ontario furniture factories was larger than respirable size. Only that dust deposited in the airways would be expected to be capable of inducing acute or chronic changes in the airways.

Pulmonary Airway Effects Not Clearly Related to Allergy

Holness et al. (8) compared the pulmonary function of 48 Ontario cabinet makers with that of 45 controls. The woodworkers had been exposed to wood dust for a mean duration of 8.3 ± 7.7 years; there was no difference in pulmonary function, as measured on the first working day of the week, between the woodworkers and controls. Over a work shift, measurements of FVC and FEV_1 among the woodworkers declined on average by about 2%, while there was no change among the controls. In contrast to these findings for lung volumes there were no significant changes in mid-expiratory flow rates over the shift. Changes of the magnitude reported for the lung volumes are unlikely to produce symptoms, and indeed, there were no significant differences in the prevalence of respiratory symptoms between woodworkers and controls.

Al Zuhair et al. (9) measured pulmonary function among 119 men at 2 English furniture factories. Total dust levels were generally well below 5 mg/m^3 . In one of the 2 factories there was an average decline in FVC and FEV_1 over the workshift of about 100 ml; in the second factory a statistically significant decline did not occur. In neither factory was there a decline in mid-expiratory flow rates. The percentage of workers with respiratory symptoms did not differ significantly from that in the normal working population.

Andersen et al. (4) measured FEV₁ and FEF₂₅₋₇₅ among 68 Danish furniture workers and found no differences from predicted values. Air sampling in the factories found 25 samples with wood dust concentrations less than 5 mg/m³ and 43 samples with concentrations greater than 5 mg/m³.

Whitehead and colleagues (10) measured the pulmonary function of 571 New England woodworkers. Three hundred and fifty individuals worked with hardwoods (mostly maple) and 221 with pine. On the basis of work history and area dust sampling, individual cumulative dust exposures were calculated and the workers were divided among high, medium, and low exposure groups. The pulmonary function test results of the workers were compared with values predicted by the Knudson (11) reference equations. For FVC there was, on average, no difference between the observed and predicted values nor was there a trend with the level of exposure. For the never-smokers, there was no difference between the observed and predicted values of FEV₁. Maximal mid-expiratory flow rates tended to be lower than predicted values. Measurements were made during the working day and one cannot determine whether these flow rates (MMEF), which are inherently a very variable measure, might reflect acute or chronic changes.

All available pulmonary function studies of woodworkers are cross-sectional in nature; as such they might underestimate a deleterious effect of wood dust exposure if severely affected workers had a tendency to leave the industry. It seems likely that woodworkers have small changes in pulmonary function over the course of a work shift which are probably related to irritation of the airways. These changes appear to be of a magnitude which are not usually associated with unpleasant symptoms and there appears to be little evidence that significant chronic changes occur.

Allergic (Asthma)

A number of woods including oak, mahogany, and western red cedar have been identified as being capable of inducing asthmatic reactions (2). The best studied of these woods is western red cedar and Chan-Yeung (12) notes that the respiratory syndrome caused by occupational exposure to red cedar is fairly characteristic. Workers with specific bronchial reactions to cedar dust had worked for many years with a variety of woods without respiratory symptoms and experienced symptoms only after steady exposure to the red cedar dust for a period of months. Nocturnal cough and wheeze were often the first symptoms. With continuing exposure, symptoms tended to occur earlier in the day. Later on, some of these patients had severe attacks of asthma immediately on exposure and constant breathlessness with effort. Upper respiratory symptoms of nasal discharge, sneezing, and obstruction were present in two thirds of the patients. A prominent feature was the absence of a previous personal or family history of allergic sensitivity. After cessation of exposure most patients recovered completely within 6 months.

All of the studies of asthma in woodworkers have been either case series or cross-sectional studies. Because workers who become ill would tend to leave the industry, it is difficult to determine true incidence rates. In a survey of cedar mill workers in British Columbia, Chan-Yeung and colleagues (13) found asthma in 1% of the cedar workers; they estimated the true incidence in this environment to be higher, around 4-5%.

There is no study which correlates the incidence of disease with dust levels. Therefore no dose-response assessment is possible.

III Cancer

Nasal Cavity and Paranasal Sinuses

During the 1960's an association was noted between employment in the furniture industry in England and an increased risk of cancer of the nasal cavity. Since then numerous international studies have confirmed the association between exposure to hardwoods in furniture manufacture and adenocarcinoma of the paranasal sinuses (14). Additional studies have found that the risk is neither limited to the furniture industry nor to hardwood exposure. Elwood (15) performed a case-control study of 121 men seen for cancer of the nasal cavity or paranasal sinuses in British Columbia between 1939 and 1977. He found increased relative risks (2.5) associated with occupations involving exposure to wood (chiefly forestry and carpentry), and with smoking (4.9). Exposure was primarily to domestic softwoods. Studies in Scandinavia (16), Australia (17), and the United States (18) confirm that exposures outside of the furniture industry (primarily to softwoods) may be associated with increased risk of sino-nasal cancer. In Ontario, Finkelstein (19) found that several deaths of sino-nasal cancer from 1973 - 1983 may be attributable to occupational exposure to wood dust. The disease is, however, so rare that wood dust exposure is probably associated with less than 1 death in the Province per year (Appendix).

The carcinogenic agent(s) in wood dust have not been identified, nor is there any data relating workplace dust levels to the risk of cancer.

Assessment of Other Sites

Acheson (20) recently reported on the follow-up of a cohort of 5108 men who had been employed in the English furniture industry prior to 1968. With the exception of nasal cancer there was no significant increase in mortality, nor any

trend towards increasing mortality with increasing dustiness of the work, for cancer of any site. Olsen and Sabroe (21) reported a 6-year follow-up of 40,000 active and retired members of the Danish carpenter and cabinet makers trade union; no major causes of death except nasal cancer were significantly above the national average. Milham (22) studied the mortality experience over 4 years of 700,000 active and retired members of the AFL-CIO United Brotherhood of Carpenters and Joiners of America. Surprisingly, the SMR for sino-nasal cancer was only 51. There was a significant excess of stomach cancer, which was more than compensated for by a significant deficit of colonic cancer. The same pattern, but not reaching statistical significance, was observed among the English furniture makers (20). The Danish study (21) found no excess of any digestive cancer.

It is concluded that the only cancers significantly associated with wood dust exposure are cancers of the nasal cavity and paranasal sinuses.

IV Occupational Exposure Limits

Occupational exposure to wood dust may be associated with dermatitis, inflammation of the eyes and nose, reversible airways obstruction and cancer of the nasal cavity and paranasal sinuses. Unfortunately there is very little data pertaining to exposure conditions and virtually none which relates the incidence of disease to dust levels. The ACGIH has recommended TLVs of 1 mg/m³ for hardwoods in furniture making and 5 mg/m³ for non-allergenic softwoods with a STEL of 10 mg/m³ for softwoods (23). The ACGIH differentiated between hardwoods and softwoods because of their impression that it was the hardwoods that were associated with sino-nasal cancer. Since their assessment, reports in the literature have suggested that individuals exposed primarily to softwoods may be at risk of sino-nasal cancer as well; thus there appears to be little justification for setting separate exposure limits for these 2 classes of woods.

In the absence of sufficient data to provide a dose/response curve for the non-carcinogenic effects of wood dust and with the evidence of a very small risk of sino-nasal cancer associated with exposure to wood dust, the selection of an upper limit of occupational exposure based on health grounds is difficult.

The following discussion reflects the sparse scientific data available for the standard setting process but attempts to approach the problem by considering this knowledge in the light of the actions of other regulatory bodies.

The Swedish occupational standard for wood dust is 4 mg/m³. There is no documentation to support this numerical standard, but their Criteria Group for Occupational Standards did examine the relationship between dust levels and upper respiratory irritation(24).

The health effects of wood dust remove it from the purely nuisance dust category to which the ACGIH assigns an upper limit of 10 mg/m³. As discussed earlier the ACGIH has assigned two exposure limit values to wood dust. The upper limit of 5 mg/m³ is based on the Danish study (4) in which the investigation found that workers exposed to wood dust above 5 mg/m³ more frequently experienced inflammation of the middle ear and that common cold symptoms, when present, lasted longer than in persons exposed to wood dust levels below 5 mg/m³.

The data relating to slowed muco-ciliary nasal transport shows that delay occurs at levels below 5 mg/m³. Although the health significance of this data is not clear, the ACGIH Threshold Value Committee agreed that impaired muco-ciliary function may be important in the development of nasal adenocarcinoma in those exposed to wood dust due to the prolonged retention of wood dust in the nasal cavity. Although the ACGIH Committee then uses this argument to set a limit for hardwoods of 1 mg/m³, they acknowledge that there is no information available on the mucostasis properties of different woods. Using this reasoning the

1 mg/m³ would be appropriate for dust derived from hardwoods if it were only this dust which was associated with the occurrence of nasal cancers. However, as shown in this document, soft woods are also associated with an increased risk of nasal cancer.

This risk is very small but where there is a risk of cancer it has been customary for regulatory bodies to select low exposure levels or the lowest exposure level reasonably achievable. Pursuing an exposure limit based on this approach and in the absence of any better scientific data the lower of the two ACGIH limits might be chosen to apply to airborne dust from all types of wood. Given the widespread and varied nature of the occupational exposure to wood dust, the final selection of an exposure limit for wood dust cannot be based on scientific criteria. Because contact with some woods can produce allergic skin reactions, procedural controls to reduce skin contact with wood and wood dust are important to reduce occupational disease in the wood handling industries.

Recommendation

It is recommended that the occupational exposure limit for wood dust be 5 mg/m³ or less.

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APPENDIX

**A Study
of
Nasal and Sinus Cancers
in Ontario:
1973 - 1983**

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There are several accepted occupational risk factors for cancer of the nasal cavity and paranasal sinuses: these include exposures to wood dust, nickel refining processes, and boot and shoe manufacturing and repair (1). This study was carried out to assess whether occupational exposure to wood dust might also be associated with an increase in mortality from these cancers in Ontario.

Ontario Statistics

In the 11 year period 1973 - 1983, 157 men 30 years of age or more were certified as dying of cancer of the nose or sinuses; this is an average of 14 deaths per year. Four percent of men were 30 - 44 years of age at death, 52% were 45 - 64, 22% were 65 - 74, and 22% were 75 years of age or older.

Not all cases of sino-nasal cancer are fatal. In the period 1973 - 1975 there were 65 new cases, an average of 22 per year (2). In 1971 the ratio of new cases to deaths was $29/18 = 1.6$ (3). The incidence rate in Ontario is thus approximately 50% higher than the mortality rate.

Materials and Methods

Deaths in Ontario for the period 1973 - 1983 receiving ICD code 160 (malignant neoplasm of nose, nasal cavities, middle ear and accessory sinuses) were identified by searching a computer tape of all Ontario deaths provided by the Ministry of Health. A control was selected for each case from all deaths in the same year, matching on age and sex, by use of random numbers. Demographic and occupational data were manually abstracted from the death certificates.

When possible, directories of Canadian manufacturers were consulted for additional information about potential occupational exposures. In a number of instances no occupational information was given on the death certificate; such data was available for 147/157 (94%) of the cases and

143/157 (91%) of the controls.

The data were analyzed using a matched-pairs technique (4); the relative risk is approximated by the A/B where A is the number of discordant pairs in which the exposed individual is the case and B is the number of pairs in which the exposed individual is the control. The relative risk is a measure of risk among exposed individuals compared with unexposed persons.

Results

a) Nickel Production

The well-known association between nickel refining at Sudbury and Port Colborne and cancers of the nasal cavity and sinuses was readily apparent; 13 cases (8% of the Ontario total) occurred among men with exposure in the nickel industry while none of the controls were identified as having been employed in this industry.

b) Wood Dust Exposure

Wood dust exposures have been divided among 5 categories; furniture and cabinetmaking; carpentry; miscellaneous industrial; pulp, paper, and forest products; miscellaneous construction. The distribution of the study population among these categories is given in Table 1. In the 11 year period there were 8 deaths of sino-nasal cancer among men with definite wood dust exposure (3 furniture workers and 5 carpenters) compared with 3 exposed controls; the relative risk of exposure is thus $8/3 = 2.7$. None of the odds ratios are formally statistically significant, but tests of statistical significance are not meaningful in this context; given the magnitude of the relative risk the sample size is too small to permit statistically significant tests.

c) Other Associations

The odds ratios for other occupational exposures are listed in Table 2. The associations with exposure to solder fumes and farming have not previously been noted and are deserving of further investigation.

Discussion

There are major limitations to a study in which occupational data is derived solely from death certificates in that there is the potential that the investigator will be unable to determine whether or not an individual has been exposed to the agent of interest and will almost certainly be unable to determine the extent of exposure. Nevertheless a study of this kind can be useful in estimating to a first approximation the magnitude of a hazard in Ontario.

There is little doubt that exposure to hardwoods in the furniture industry is associated with sino-nasal cancer (5) and a recent study from British Columbia suggests that the risk is present with exposure to domestic softwoods as well (6). Three men dying of sino-nasal cancer in Ontario during 1973 - 1983 were cabinetmakers or furniture workers; one was born in Hungary and another in England and they may have had wood dust exposure outside of Canada. Five carpenters died of sino-nasal cancer. No cases had a forest industry occupation specified although it is possible that some men said to be labourers (not otherwise specified) may have been employed in the forest industry or some men may have worked in this industry earlier in life. Two cases and 2 controls had employment in the pulp and paper industry. Construction workers were more represented among the cases than among the controls, but any association with wood dust exposure is quite tenuous.

Conclusions

It is probable that several deaths in Ontario in the period 1973 - 1983 are attributable to exposure to wood dust; it is also possible that several non-fatal cases of sino-nasal cancer may be attributable to this exposure. Nevertheless sino-nasal cancer remains a very rare cause of death and it is likely that on average less than 1 death per year in the Province of Ontario is attributable to occupational exposure to wood dust.

Acknowledgments

Bob Kusiak identified the cases and selected the controls.
Jackie McPherson abstracted the death certificate data.

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TABLE 1 The Distribution of Wood Dust Exposures Among the Study Population

<u>Cases</u>		<u>Controls</u>	<u>Odds Ratio</u>
I <u>Furniture and Cabinet Making</u>			
Cabinetmaker	Age 37	Furniture Restorer	3
Cabinetmaker	Age 81		
Furniture Worker	Age 64		
II <u>Carpentry</u>			
Carpenter	Age 71	Carpenter	2.5
Carpenter's Helper	Age 47	Carpenter	
Carpenter	Age 62		
Carpenter	Age 45		
Carpenter - Industrial	Age 76		
III <u>Miscellaneous Industrial</u>			
		Mechanic and Engineer -	
		- Brush Manufacture	
		Labourer - Box Manufacture	
IV <u>Pulp and Paper</u>			
Paper Mill	Age 58	Labourer - Pulp	1.0
Supervisor	Age 50	Blacksmith - Paper Mill	
V <u>Miscellaneous Construction</u>			
Builder - Government	Age 80	Construction	3
Labourer - Construction	Age 50	Roofer	
Self-Employed Contractor	Age 48		
Bricklayer	Age 45		
Truck Driver - Construction			
	Age 66		
Crane Operator - Construction			
	Age 48		

Table 2

Other Associations
Between Occupation and Sino-Nasal Cancer

<u>Exposure</u>	<u>Cases</u>	<u>Controls</u>	<u>Relative Risk</u>
Nickel Manufacture	13	0	---
Leather/Shoes	3	2	1.5
Farming	13	7	1.9
Solder Fumes	8	1	8
Textiles	1	1	1